HEARING LOSS AND CORONARY HEART DISEASE*

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Our first study in 1960-61[1] among members of the Mabaan tribe in southeast Sudan revealed their hearing at 500 to 6000 cps. to be significantly more acute in all decades from 10 through 70 years than in industrial areas of the United States[2] (Figure 1). Except for the bleat of a goat and other sounds of nature, the Mabaans live in a dramatically quiet, almost silent atmosphere. The bombardment of excessive noise in our culture and the virtual absence of such in theirs could be one of the factors responsible for their superior hearing.

Generally, hearing loss and increase in blood pressure occur with aging in healthy persons of the United States,[3] while the Mabaans' systolic and diastolic blood pressures remain the same at 75 as at 15 years of age, and coronary heart disease is unknown in this tribe (Figures 2 and 3). The Mabaans probably have minimal generalized atherosclerosis and greater elasticity of the small arteries.[4] We saw no varicose veins or thrombosis, no bronchial asthma, duodenal ulcer, nor ulcerative colitis. Rheumatic heart disease was not found among the children. The Mabaans are well nourished, their posture is erect at all ages, the body musculature is well developed and firm. We saw no obesity. They seem to age more slowly and live longer than we do and remain agile in their seventies and eighties.

Their diet is frugal. The main food is a ground millet, which they eat as a wet mash and from which they also make beer. They eat fish, nuts, and wild dates. They possess a few scrawny cattle, a few pigs and

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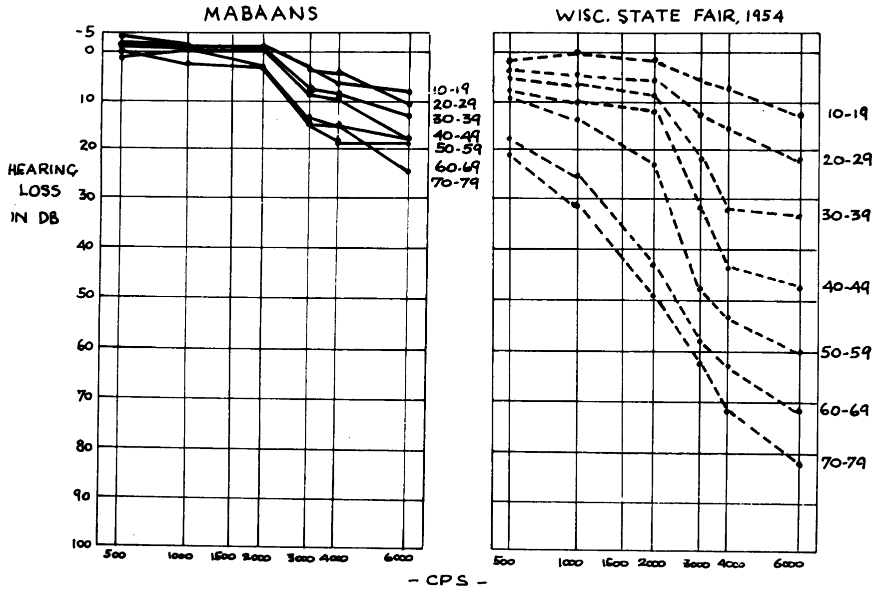

Fig. 1. Comparison of decade audiograms in men (medians used).

goats that are never slaughtered intentionally but are eaten as they die. A few small eggs are available and are given to the youngest children. Their mean average cholesterol is 160 mg. per cent in contrast to ours of 250 mg. per cent. They are physically very active, especially the women, who perform the heaviest work, carrying firewood and water on their heads for long distances. The Mabaans do not smoke. In two separate studies with our group Dr. T. A. Baasher[5] of The Clinic for Nervous Disorders, in Khartoum, Sudan, and senior psychiatrist of the Republic of the Sudan assessed their lives to be singularly free of "stress" as we know it. There is, however, always the real fear of wild animals and poisonous snakes, and concern over whether the millet crops will last through the rainy season. Acute hearing is necessary for survival, so they have learned to listen since early childhood.

Occasionally when a few Mabaans leave their area to live in the big city—Khartoum—they become prone to hypertension, hypercholesteremia, coronary heart disease, and the stresses incident to big-city life.

High-frequency (12 to 24 kc) studies,[6] conducted in 1962, again revealed the Mabaans' hearing acuity to be far greater than that of similar age groups in the city of New York, in Düsseldorf, Germany, and in Cairo, Egypt (Figure 4). In 1961 Glorig and Davis[7] called our

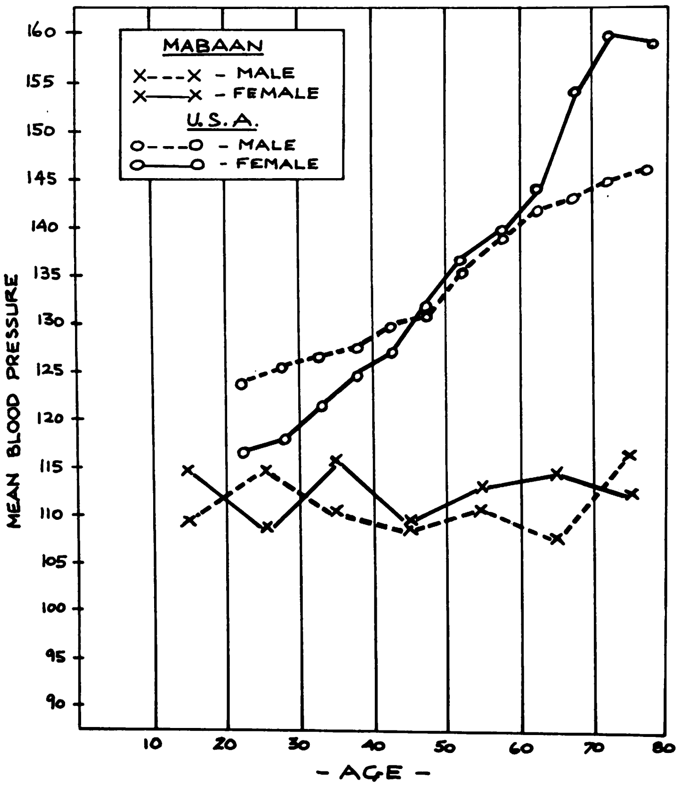

Fig. 2. Comparison of mean systolic blood pressure of apparently healthy persons in the U.S.[3] and the Mabaan Tribe, Sudan.

attention to an air-bone gap with aging and reported a 12-db air-bone gap at 4000 cps. at age 55 in healthy persons in the United States who had not been exposed to noise (Figure 5). This is the typical air-bone gap seen in conductive loss. Glorig and Davis believed this gap resulted from loss of elasticity of the tissues of the ossicular joints because of the infiltration of an increased amount of connective tissue generally found throughout the body with aging. They thought similar changes in the basilar membrane caused it to stiffen and that this also interfered with sound conduction. In 1963[8] we measured the air-bone gap of the Mabaans. This study revealed that they too have an air-bone gap at age 55, but their gap is 50 per cent less than ours. The Mabaans do not reach the 12-db air-bone gap of the 55-year-old citizens of the United States until the age of 75. Their biological time clock runs more slowly than ours, hence their connective-tissue changes occur later than ours.

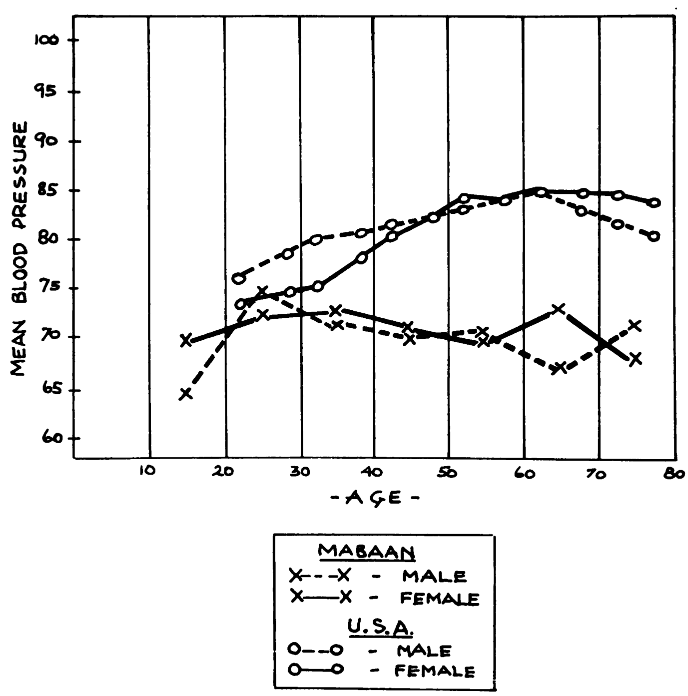

Fig. 3. Comparison of mean diastolic blood pressure of apparently healthy persons in the U.S.[3] and the Mabaan Tribe, Sudan.

It seemed doubtful that the absence of noise alone could account for the superior hearing of the Mabaans. We therefore began to study hearing loss and air-bone gaps in populations where the diet, cholesterol levels, blood pressures, incidence of cardiovascular and coronary heart disease, exercise, smoking, etc., were in marked contrast to these factors among the Mabaans. This report covers our study of March and April, 1964, in Finland, a country with one of the highest saturated fat diets and one of the highest incidences of coronary heart disease in the world.

In the last ten years research has shown correlations between dietary fats, blood lipids, and coronary heart disease. From this evidence a hypothesis has emerged, according to which a high content of saturated fats in the diet would disturb the normal content and composition of blood lipids, and this, in turn, would accelerate the development of atherosclerosis and thrombotic phenomena, leading to increased incidence of coronary heart disease. Thus coronary heart disease in one way could be considered a nutritional disease.[9-11]

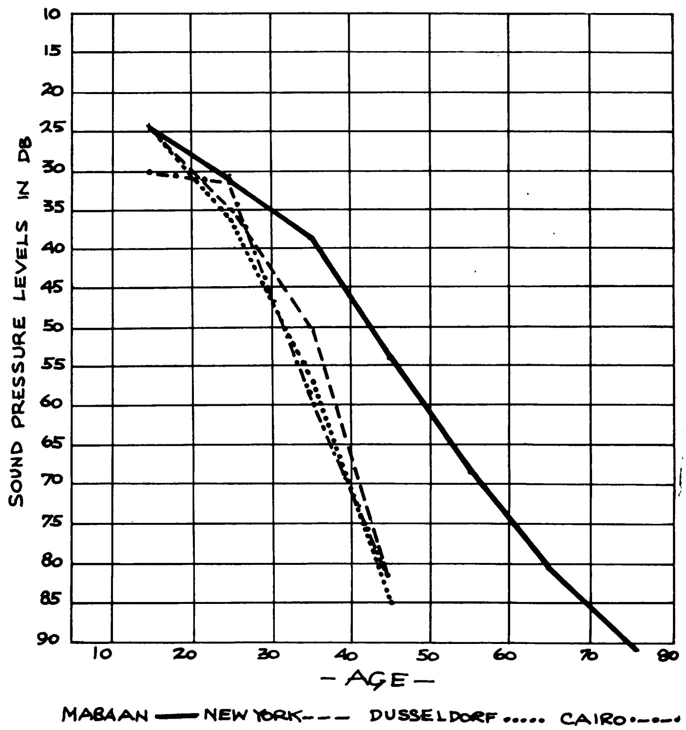

Fig. 4. Comparison of decade audiograms (medians: 14 kc).

Could this disease, whatever its mechanism, bear a relation to hearing loss? For many reasons, and not the least from the point of view of preventive medicine, it would be of utmost importance either to prove or disprove this lipid hypothesis. This can be done, it would seem, by means of adequately planned direct experiments on humans in which the diet of a suitable population is changed to satisfy the requirements of the hypothesis and by observing the development of manifestations of coronary heart disease over a sufficiently long period. An otherwise comparable population among whom no dietary change is instituted is required as a control. Following a preliminary study,[12] such an experiment was begun five years ago by Turpeinen, Karvonen, and Roine in Finland, and in a paper titled "Dietary Prevention of Coronary Heart Disease" read at the Sixth International Congress of Nutrition in Edinburgh, Scotland, August 14, 1963, they reported their observations of the first three years of this experiment on 498 males from 35 to 64 years of age. This experiment is still in process; the final findings of this six-year study will be published soon.

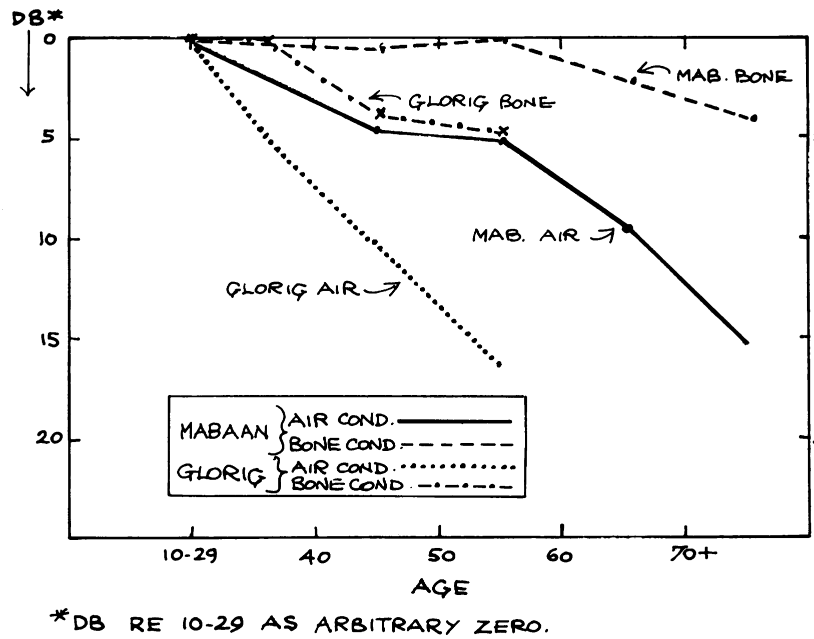

Fig. 5. Air-bone gap (4 kc) Mabaan and Glorig.

The experiment is being carried out in two mental hospitals in the vicinity of Helsinki, with 1000 and 640 patients, respectively, mostly asylum cases. The type of patient and treatment in both hospitals were not significantly different. The patients live under uniform environmental conditions and are in hospital residence from 1 to 25 years or more.

In one of them, designated here as Experimental Hospital, whose patients come from the city of Helsinki, the diet was changed so that a large part of the saturated fat was replaced by soybean oil, whereas in the other hospital, here designated Control Hospital, the patients, who come from rural areas, were kept on the usual Finnish diet. Repeated dietary studies, serum cholesterol determinations, and electrocardiographic examinations were carried out in both hospitals.

Before the start of the experiment the diets in both hospitals had been similar and typical of Finnish dietaries in that they contained large quantities of saturated fat derived mainly from whole milk and butter. At the start, the diet in the Experimental Hospital was changed mainly in two respects. First, whole milk was replaced by an emulsion of soybean oil in skim milk. Second, butter and margarine were re-

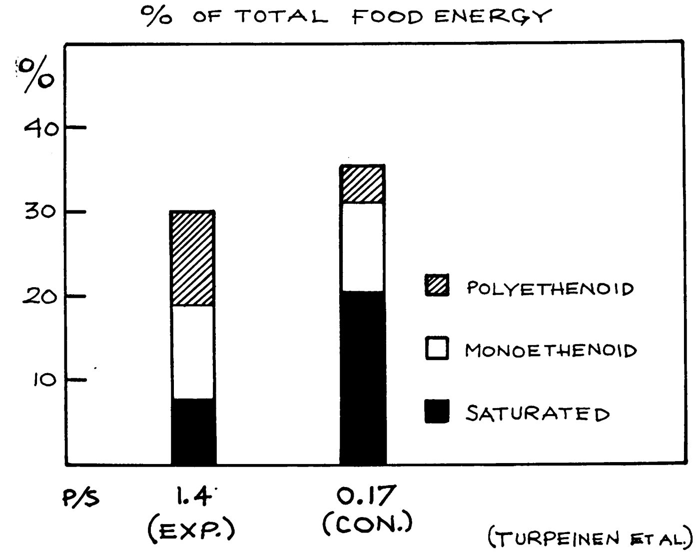

Fig. 6. Fatty acid content of the diets in Experimental Hospital and Control Hospital.

placed by a special brand of margarine that contained considerably more unsaturated fat than any of the brands marketed in Finland. The main effect of these dietary changes was that the milk fat present in the original diet was almost totally replaced by vegetable oils, mainly soybean oil. After this change the fatty acid compositions in the two hospitals were greatly different, as shown in Figure 6. In the Experimental Hospital the diet contained much less saturated acids and much more polyunsaturated acids than in the Control Hospital. Hence the P/S ratios were very different.

The mean serum cholesterol levels of the subjects in each of the two hospitals are shown in Figure 7. The initial cholesterol level was slightly higher in the Control Hospital. This was thought to be due to the somewhat higher total content of dietary fat in that institution. The change of the diet, however, caused a marked fall in the cholesterol in the Experimental Hospital. No such change occurred in the Control Hospital. Actually, there has been a slight tendency toward a progressive rise of serum cholesterol in this hospital; hence the difference between the two hospitals tended to increase. After three years of diet, the mean cholesterol level in the Experimental Hospital was 220 mg. per cent and in the Control Hospital 267 mg. per cent. The differences in the cholesterol levels have been statistically significant at all times after the start of the experiment.

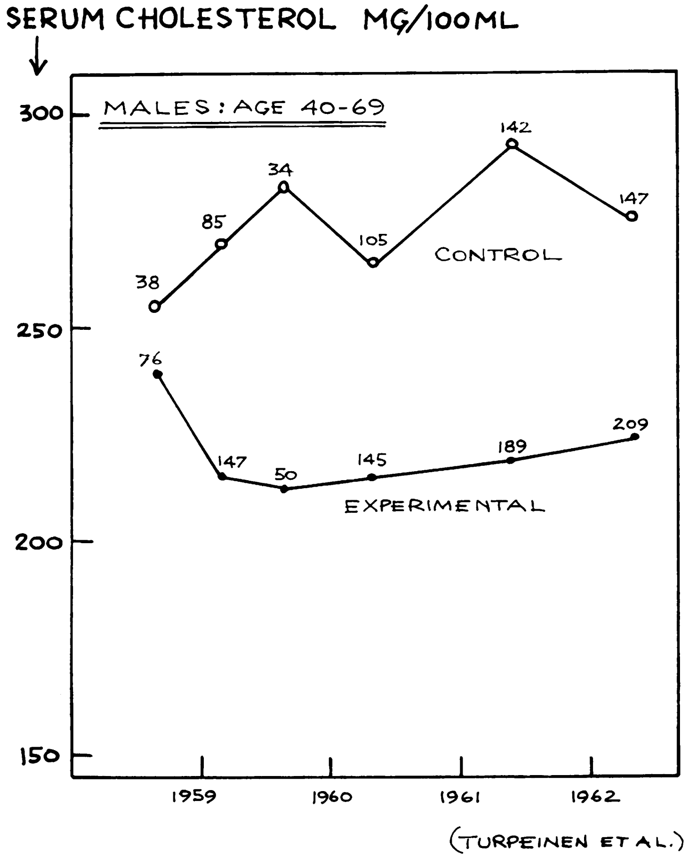

Fig. 7.

The dietary change also affected blood coagulability, measured as the whole blood coagulation time. This is shown in Figure 8. Initially the mean coagulation times in both hospitals were identical (clear columns), but six months after the change of diet, this time had significantly lengthened in the Experimental Hospital, whereas no change was observed in the Control Hospital. This phase of the work was carried out in collaboration with Dr. Ratko Buzina, of the Department of Nutrition, Institute of Public Health, Zagreb, Yugoslavia.

Though all abnormalities in electrocardiography were noted, special attention was paid to the changes held indicative of myocardial ischemia. The prevalence of these abnormalities is shown in Figure 9. At the outset their prevalence was the same, that is, they occurred in 7 per cent of all electrocardiograms examined in each of the two hospitals,

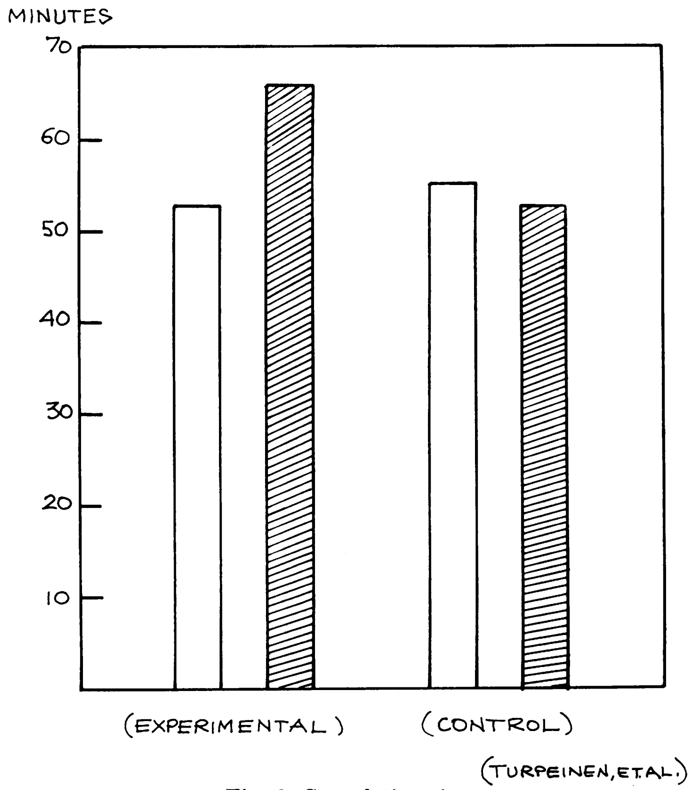

Fig. 8. Coagulation time.

increasing to about 12 per cent in the Experimental Hospital and to 15 per cent in the Control Hospital. The authors state that there appears to be a slightly lower incidence in the Experimental Hospital, but the figures are too small for statistical validity.

They assessed the electrocardiographic findings and the mortality data on the incidence of *new* coronary heart disease, that is, all deaths due to new coronary heart disease and all *new* electrocardiographic abnormalities indicating either "probable" or "possible" myocardial infarction. Table I shows the differences in the two hospitals. In all of these cases there was no previous electrocardiographic abnormality.

The cases of new coronary heart disease have been definitely less numerous in the Experimental Hospital, in which the diet was changed, than in the Control Hospital. As shown by the numerical value of X^2 and of P, the difference is quite significant. Again the authors stated that the total number of cases is so small that one would still remain a little hesitant to draw final conclusions. Nevertheless, these observa-

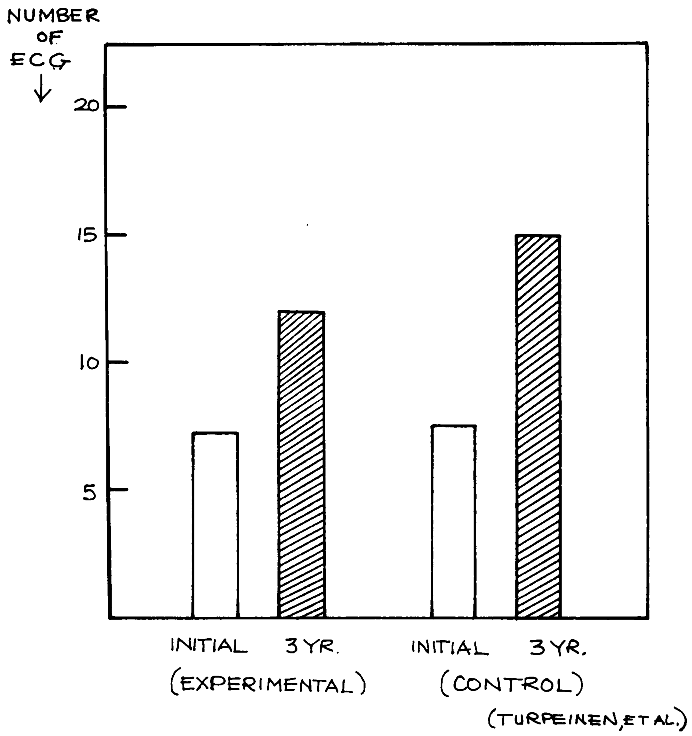

Fig. 9. Evidence for myocardial ischemia per 100 ECG's.

tions of Turpeinen, Karvonen, and Roine led them to the preliminary conclusion that a high-saturated fat diet favors the development of coronary heart disease.

Five years after the beginning of the dietary experiment, we performed hearing studies in these two hospitals, testing 136 patients in the Experimental Hospital and 142 patients in the Control Hospital. Three of us did the testing: Helen V. Rosen and ourselves. The patients were tested in matched age groups and on their ability to provide reliable, consistent responses to stimuli. The audiometric data obtained on each patient consisted of air-conduction tests at 500-4000 cps., bone-conduction tests at 2000 cps. and 4000 cps., and high-frequency tests at 12 to 24 kc. In the analysis of data, four groups resulted. In the Experimental Hospital, one group aged 40 to 49 (39 cases) and another group, aged 50 to 59 (97 cases); in the Control Hospital, one group aged 40 to 49 (37 cases) and another group aged 50 to 59 (106 cases).

Figure 10 shows air-conduction means for each of the four groups.

TABLE I—THE DEVELOPMENT OF NEW CASES OF CORONARY HEART DISEASE AMONG PATIENTS WITHOUT PREVIOUS ECG ABNORMALITIES

	New CHD	*No new CHD*	*Total*
Experimental	1	221	222
Control	6	156	162
Total	7	377	384

$X^2 = 5.54$ $n = 1$ $P < 0.02$

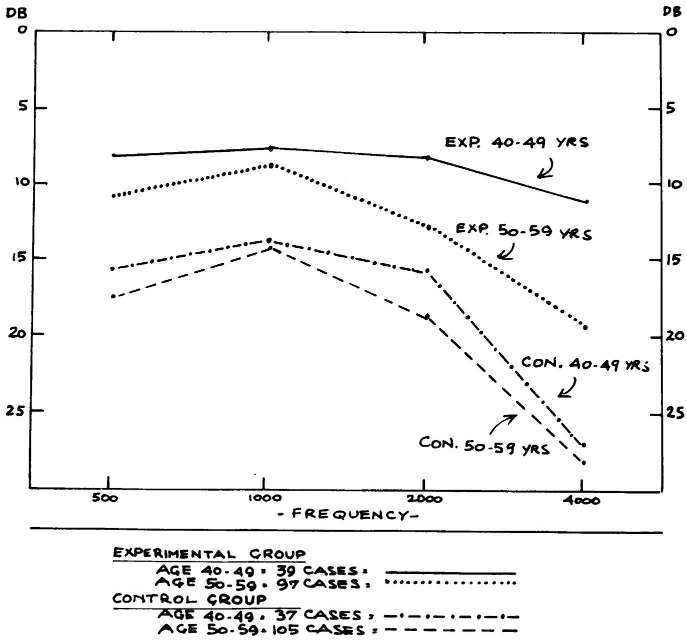

Fig. 10. Air conduction means (Finland).

As may be seen, the means for the Experimental Hospital regardless of age are better throughout the entire audiometric range than those of the Control Hospital. Patients aged 50 to 59 in the experimental group have better hearing than those 10 years younger in the control group. Further age-group comparisons for significant differences were made between the experimental and control mean of the individual frequencies. It was found that the mean in each individual frequency in the Experimental Hospital was significantly better than the corresponding mean in the Control Hospital. In each comparison the "t" value was

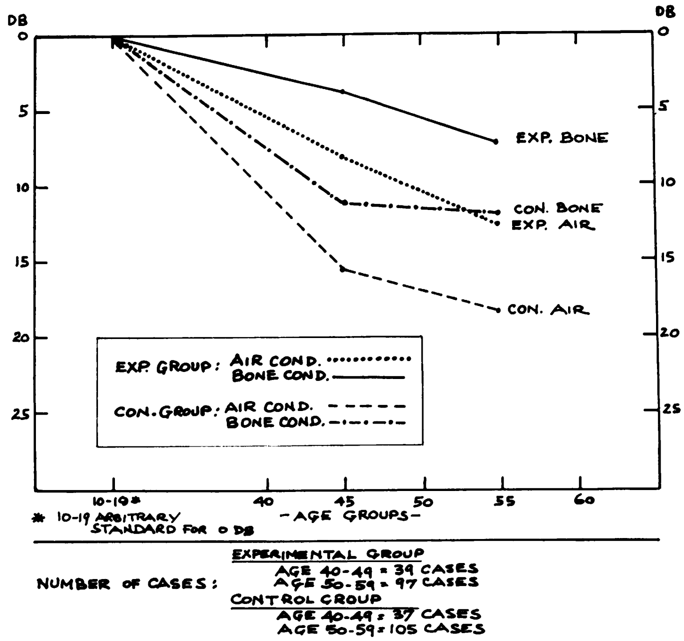

Fig. 11. Air-bone gap at 2000 cps. (Finland).

well over 2.5 and thus significant to levels of 0.005 or better. Figure 11 shows the air-bone gap of each age group at 2000 cps. The differences in the means at 2000 cps. between the corresponding age groups in the two hospitals are not statistically significant. At 2000 cps. the air-bone gap in the study of Glorig and Davis was likewise not statistically significant.

At 4000 cps. (Figure 12), the air-bone gap in the Experimental Hospital is much smaller than that of the corresponding age group in the Control Hospital. The gap at age 40 to 49 in the Experimental Hospital is 1.4 db whereas in the Control Hospital it is 10.5 db. The gap at age 50 to 59 is 4.1 db in the Experimental Hospital and 9.9 in the Control. These great differences in air-bone gaps between hospitals give "t" values over 4.0 and thus are highly significant to levels of 0.00003.

Medians of the four groups have been found for the high frequencies 12 to 24 kc (Table II). Again, hearing at 12 kc in the older age group (50 to 59) of the Experimental Hospital is as good as the 10-year-younger age group (40 to 49) of the Control Hospital. Also, the percentage of response to the stimulus in the Experimental Hospital

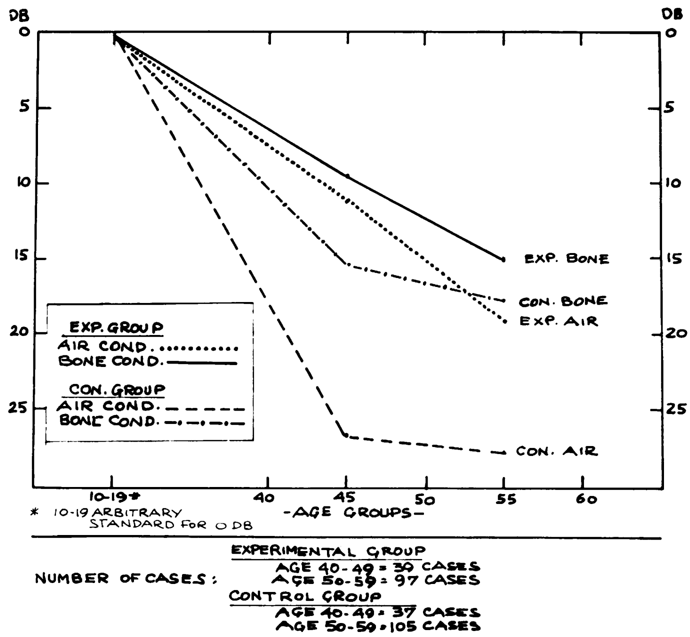

Fig. 12. Air-bone gap at 4000 cps. (Finland).

is higher than that of the corresponding age group in the Control Hospital.

Table III shows these same high-frequency medians in relation to those obtained in the Mabaan tribe and in the United States. It is interesting to see how much more superior the Mabaans' high-frequency hearing is as compared to ours and, again, how much more superior these two groups are as compared to either Finnish hospital. This applies to medians and to the percentage of response of each frequency.

In résumé, the Experimental Hospital patients, aged 50 to 59, heard 500 to 4000 cps. by air conduction better than patients aged 40 to 49 in the Control Hospital. At 12 kc in the Experimental Hospital the 50- to 59-year-old patients heard as well as the 40- to 49-year-old patients in the Control Hospital. If the Glorig and Davis explanation of the conductive air-bone gap as a gradual diminution of elasticity of tissue inherent in aging is correct, then the much smaller air-bone gap at 4000 cps. in the Experimental Hospital would again suggest that these patients age more slowly than those in the Control Hospital.

Just what is the effect of this apparent accelerated aging process in the patients in the Control Hospital? One of the principal structural

TABLE II—MEDIANS (HIGH FREQUENCY: 12 to 24 KC). Finland

	Age 40-49		*Age 50-59*	
	Exp.	*Con.*	*Exp.*	*Con.*
12 kc	70.00 db 85%	87.50 db 62%	88.75 db 59%	MNO 42%
14 kc	92.50 db 51%	MNO 38%	MNO 19%	MNO 12%
16 kc	MNO 10%	MNO 3%	NR 0%	NR 0%
18-24 kc	NR 0%	NR 0%	NR 0%	NR 0%
No.	39	37	97	105

MNO = Median not obtainable.
NR = No response.

effects seems to appear in the cardiovascular system, giving rise to generalized atherosclerosis and coronary heart disease. This study shows that the difference in hearing in the two hospitals parallels the difference in the incidence of coronary heart disease. One must wait for the further passage of time and, with it, the occurrence of more cases of coronary heart disease to confirm this association. Therefore it is very important to have autopsy evidence in these cases as to the state of the carotid and vertebral arteries and also the arterial and capillary blood supply to the inner ear, since diminished blood supply could alter cochlear function.

At what age does the long incubation period in the pathogenesis of atherosclerosis and coronary heart disease begin? If our vascular theory of hearing loss is plausible, then young people (10 to 29 years) in a population where there is a high incidence of coronary heart disease should have less acute hearing for the high frequencies (12 to 24 kc) than a similar young-age group in a population area where the incidence of coronary heart disease is low.

Pursuing this final question we recently made a pilot study of two such groups of young people (10 to 29)—one in a remote area of East Finland near the border of the Soviet Union and the other in the mountains on the Dalmatian coast of Yugoslavia. The 40- to 59-year-old population in East Finland is notoriously hypercholesteremic (mean 297 mg. per cent) and has the highest incidence of coronary heart

TABLE III—COMPARISON OF MEDIANS (HIGH FREQUENCY: 12 to 24 KC)

	Age 40-49				*Age 50-59*			
	Mabaan	*U.S.A.*	*Exp. Hosp.*	*Con. Hosp.*	*Mabaan*	*U.S.A.*	*Exp. Hosp.*	*Con. Hosp.*
12 KC	35.0 DB 100%	50.7 DB 95%	70.0 DB 85%	87.5 DB 62%	53.6 DB 98%	77.9 DB 82%	88.8 DB 59%	MNO 42%
14 KC	55.0 DB 94%	83.7 DB 70%	92.5 DB 51%	MNO 38%	67.5 DB 86%	MNO 38%	MNO 19%	MNO 12%
16 KC	89.6 DB 57%	MNO 28%	MNO 10%	MNO 3%	MNO 31%	MNO 3%	NR 0%	NR 0%
18 KC	MNO 18%	MNO 7%	NR 0%	NR 0%	MNO 8%	NR 0%		
20 KC	MNO 4%	NR 0%			MNO 2%			
22 KC	MNO .9%				NR 0%			
24 KC	MNO .9%							
No.	108	105	39	37	108	117	97	105

MNO = Median not obtainable.
NR = No response.

TABLE IV—PERCENTAGE RESPONDING, WITH MEDIANS, AT 12 TO 20 KC, OF THE TOTAL NUMBER TESTED IN DALMATIA, YUGOSLAVIA, COMPARED TO EAST FINLAND

	12 KC		*14 KC*		*16 KC*		*18 KC*		*20 KC*	
Age 10-19	*%*	*Med.*	*%*	*Med.*	*%*	*Med.*	*%*	*Med.*	*%*	*Med.*
Dalmatia (46 cases)	100	27.1	100	34.1	96	48.8	87	75.5	35	MNO
East Finland (21 cases)	100	25.0	100	38.1	95	46.3	71	82.5*	14	MNO
Age 20-29										
Dalmatia (22 cases)	100	32.0	100	45.8	91	70.8	46	MNO	5	MNO
East Finland (21 cases)	95	30.5	95	55.0*	71	85.0*	10	MNO	0	MNO

MNO = Median not obtainable
* = Statistically signficant

disease in Finland, whereas the 40- to 59-year-old Yugoslavs have a much lower mean blood cholesterol level (183 mg. per cent) and one of the lowest rates of coronary heart disease in all of Europe.

The young Yugoslavs hear the high frequencies better than the young Finns. In the 10- to 19-year range, the Yugoslavs hear better at 18 kc., and in the 20- to 29-year-old group, the Yugoslavs hear better at 14, 16, and 18 kc (Table IV). Does the poorer hearing of the Finns in the age range of 10 to 29 years reflect the beginning of the long pathogenic vascular process that may eventuate in atherosclerosis and coronary heart disease?

Enos, Holmes, and Boyer[13] dissected the coronary arteries of 300 soldiers killed in action in Korea. The average age was 22.1 years. They found gross evidence of coronary disease in 77.3 per cent of the cases. The disease varied from "fibrous" thickening to complete occlusion of one or more of the main branches.

It is at this point that the otologist may be able to make a contribution to the increasing data being gathered on the chief enemies—atherosclerosis and coronary heart disease. Pursuing our research further, as we are presently doing, we may be able to say, as otologists, that the preventive treatment of atherosclerosis must begin long before the evidence of diminished hearing of the high frequencies can be observed. It is not enough for the 40-year-old man, conscious of his expanding waistline and the coronary deaths of his friends, to begin a regime based on polyunsaturated fats and daily exercise and moderation of work, play, ambitious goals, etc. It is perhaps prudent to begin this educational process with young children. Many cardiologists have suggested the hypothesis that diet control should begin early in life. Our studies, showing the early signs of cochlear changes in children and young adults, point an otological finger to the value of this hypothesis.

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